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EDITORIAL

MILK POLICY

On July 8, following announcements in both Houses of Parliament, there was published by H.M. Stationery Office the White Paper (Cmd. 6454) on "Measures to Improve the Quality of the Nation's Milk Supply." The changes envisaged will give much satisfaction to all interested in the subject and will go far to ensure the provision of better and safer milk. Two main methods to obtain the necessary improvement are discussed—one concerns the Minister of Food and the other the Minister of Agriculture. The Minister of Food, it is proposed, will have powers to make it an offence to sell milk by retail in any area which he may schedule "unless it is heat-treated as defined by Order, or is lawfully sold as T.T. milk, or it is Accredited milk sold by a retailer (whether producer-retailer or dairyman) who sells the milk of a single Accredited herd."

The Minister of Agriculture will become responsible for milk production. Attention is drawn to the improvements in the quality of milk likely to follow the maintaining of well-bred, healthy herds, and great importance is attached to the rearing and use of better bulls, the culling of unsatisfactory and unhealthy cattle, and the general grading-up of dairy herds throughout the country. The veterinary inspection of herds will be reorganised so that every herd will be inspected at least once a year. This may seem a small contribution to the ensuring of healthy cattle, but "it will provide for the first time a uniform

basis of inspection of dairy herds throughout the country and bring the State veterinary officers into regular contact with those farmers (owning 'non-designated' herds) who most need their advice. It is anticipated that an additional 30,000 inspections per year will be made. Although every herd will be inspected once a year, some herds will be examined more frequently. Thus, T.T. herds will continue to be inspected every six months as at present; Accredited herds will be inspected at least once a year where the milk is subject to adequate heat-treatment before sale to the consumer; where milk from an Accredited herd is not so treated the herd will continue to be inspected once every three months, except the herd is Attested, in which case it will be inspected once in every six months. All other herds, the milk from which is heat-treated, will be inspected once a year." Reference is also made in the Paper to "the scheme for the control of diseases of dairy cattle recently introduced under the auspices of the National Farmers' Union and the National Veterinary Medical Association, with the approval of and some financial assistance from the Ministry of Agriculture." It is stated that "every effort will be made, particularly by inspectors carrying out routine inspections, to encourage an extension of this scheme within the limits imposed by the availability of veterinary practitioners in any particular district."

To encourage farmers to produce more milk free from tubercle infection it is proposed to change the present basis of remuneration and to pay a premium of fourpence a gallon direct to the producers. In addition, the Ministry of Food will take over the responsibility of finding a market for T.T. milk and, in order to encourage its consumption throughout the country, will arrange that it is sold to the public at a price only slightly in excess of that for ordinary milk (raw or heat-treated).

It is recognised that, in order to obtain satisfactory milk, the conditions under which it is produced require consideration; cowsheds, dairies, etc., must receive proper attention. It is proposed that "the Minister of Agriculture and Fisheries will . . . become responsible for all matters relating to the production of milk," and it is further stated that "the veterinary staff of the Department, who will in any case be visiting the farm at regular intervals to inspect the animals, will then also be responsible for the supervision of the conditions under which the milk is produced. Under central control it will be possible to bring about greater uniformity of standard and a continuous improvement in that standard." This means a transfer of existing powers of local authorities to a central authority and, following consultation with local authorities concerned, it is proposed to submit to Parliament "legislation providing for the transfer to the Ministry of Agriculture and Fisheries, as soon as conditions permit, of the functions of local authorities relating to the conditions under which milk, including designated milk, is produced on the farm." This represents a step of much importance and will ensure that those so intimately concerned with the health of livestock will be able to use their special training and experience in providing the conditions most likely to maintain healthy cattle and clean, wholesome milk.

It is to be noted that all the policy as outlined for England and Wales

will not be applied to Scotland. It is not considered necessary, for example, to transfer the functions in regard to the inspection of dairy premises from the local authorities to a central authority; the requiring of heat-treatment as above and the new premium for T.T. milk will, however, apply.

As far as Northern Ireland is concerned, certain adjustments will be necessary, largely because of the difference in the conditions regarding the grading of the milk and the methods of licensing and inspection both of dairy cows and of dairy premises.

It cannot be expected that the various proposals can be brought into being in the immediate future: time will be necessary to take over the new duties and to arrange for the increase of veterinary work involved. This work will mean that the Veterinary Profession will be called upon to a greater extent and that preventive medicine will be practised more in the future than in the past. This is a position we have envisaged: it is the duty of every member of the Profession called upon to participate in the scheme to apply himself to the best of his ability and so to ensure its success.

RETIREMENTS

We note that announcements have recently been made of the retirement of two outstanding veterinarians.

In *Veterinary Medicine* (June issue) reference is made to the retirement of Dr. John R. Mohler, Chief of the Bureau of Animal Industry, United States Department of Agriculture. Dr. Mohler has held this post since 1917 and has spent altogether forty-six years in the service of the Bureau, passing through different posts, including those of assistant pathologist, zoologist, chief of the Pathological Division and assistant chief of the Bureau. Dr. Mohler will be remembered for his many activities, including his literary contributions, of which, perhaps, the best known in this country is his work on the English editions of Hutyra and Maret's Special Pathology and Therapeutics of the Diseases of Domestic Animals. In 1930 he was made an Honorary Associate of the Royal College of Veterinary Surgeons. Many of our readers will recall his presence at the last International Veterinary Congress in London when he read some interesting papers.

It is reported in the *Canadian Journal of Comparative Medicine* (April issue) that Dr. A. E. Cameron has retired from the post of Veterinary Director General of Canada, Health of Animals Division, Dominion Department of Agriculture. After a varied career in different parts of the world following his leaving his native land of Scotland, he graduated in veterinary medicine from the Ontario Veterinary College and soon afterwards joined the Health of Animals Branch. Subsequently he occupied various posts, and in 1925 became Chief Veterinary Inspector. In 1939 he succeeded Dr. George Hilton as Veterinary Director General.

To both we extend our good wishes for a full measure of enjoyment in their well-earned retirements.

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General Articles

SPIROCHÆTOSIS OF FOWLS IN INDIA—I

By M. K. SREENIVASAN and N. S. SANKARANARAYAN
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Contribution to the Study of Fowl Spirochætosis

SINCE the discovery of *Spirochæta anserina* by Sakharoff (1891) extensive work has been carried out on fowl spirochætosis in various countries, particularly in those of the southern hemisphere, where the disease seems to be more prevalent than elsewhere. In spite of its economic importance, the disease has up to the present escaped systematic investigation. Foster Reaney (1907) was the first to report the occurrence of spirochætosis in Central India. Montgomery (1908) met the disease in the North-western Punjab and in the United Provinces. He incriminated the tick, *Argas persicus*, as the transmitting vector. The next important paper reporting investigations upon this disease is that by Knowles, Das Gupta and Basu (1932). These authors give a very extensive review of the literature. Their report, however, is mainly of academic and entomological interest and the field veterinarian is left in the dark as to the treatment of individual cases and the carrying out of preventive measures on poultry farms.

The study of the disease, as may be seen from the literature, is far from being concluded. Although the reports correctly describe one or other outstanding feature of the disease, many of the statements contradict one another and some have not been confirmed by further work. The purpose of this paper, therefore, is to record the writers' observations on the natural and artificial disease, and certain experiments directed towards its control.

On the subject of nomenclature, Sakharoff (1891) who first discovered the parasite in geese in Caucasia, named it *Spirochæta anserina*. Marchoux and Salimbeni (1903) found the disease among fowls in Rio de Janeiro and they are of opinion that the Brazilian parasite is different from the Caucasian variety. According to Sakharoff, *Sp. anserina* is incapable of infecting chickens, while *Sp. gallinarum* can infect not only chickens but geese, rabbits and sparrows. Nuttall (1913) says that *Sp. anserina* is synonymous with *Sp. gallinarum*. Stephens and Christophers (1904) were the first to use the name, *Sp. gallinarum*. Wenyon (1926) considers that the various spirochætes described from fowls, geese and ducks belong to one species and that the correct name for the spirochæte is *Treponema anserinum*, after Sakharoff. However, Wenyon admits that spirochætosis in fowls has now been found to occur in many parts of the world, though the disease in geese has been less commonly seen. Thomson and Robertson (1929), on the other hand, have described the parasites in geese, fowls and ducks as *Sp. anserinum, gallinarum* and *anatis*, respectively. Beyond naming them separately no differentiation has been mentioned. The strain concerned in the present review was originally isolated from a fowl, but it produced in ducks a disease with identical severity and symptoms. It is interesting to note, however, that when two clean fowls were subinoculated from these ducks at the height of infection, the former manifested practically no clinical symptoms, showing only a few parasites in the blood for two days and finally recovering.

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Since the disease is of more importance in fowls than in geese, it appears reasonable to use the name *Sp. gallinarum* for the parasite concerned.

Cantacuzéne (1899) reports that adult fowls are almost immune, and that young chickens are most susceptible, with death occurring as a rule within twenty-four hours of the first appearance of spirochætes in the blood. He found that organisms in blood taken at the height of infection die out in half an hour, while spirochætes in blood taken at the end of lysis may survive *in vitro* for two or three days. From this, he infers that lysins are produced in the shed blood. It is now known, however, that adult birds are as susceptible as chickens and that death in almost all cases occurs only at the crisis. The writers have found living and viable spirochætes in samples kept for as long as ninety-seven days in the refrigerator, while in vaselined preparations made at the height of infection the parasites have been found living even after twenty-four hours at Mukteswar (7,500 feet). Hindle (1911) reports that in infected blood in sealed tubes the spirochætes live for three months. Neiva (1914) kept them at 0° for forty-nine days and found them virulent.

Balfour (1907, 1908) describes certain bodies in the red cells as stages in the after-phase of fowl spirochætosis. In a later report (1914) he mentions them as precursors. In 1913 he queries his previous theory and suggests that, if the granules are not some phase of the parasite, they are possibly chlamydozoal in character and transmitted by ticks. Gleitsmann (1913), however, believes the granules to be degeneration products formed by toxic action on erythrocytes and this is supported by Knowles *et al.* (1932), who produced them in the blood by injecting phenol products. Balfour (1914) experimented with the granules (inclusion bodies) by infecting chickens with tick eggs and finally concludes that the granules may be some new parasites closely associated with spirochætes and having a common vector in *A. persicus*. Curasson and Andrejesky (1929) made an extensive study of "Balfour's bodies" and spirochætes and proved conclusively that the two represent different parasites. Marchoux and Couvy (1912, 1913), continuing the experiments of Balfour, considered such granules to be normal structures in a wide variety of acarines, having no connection with spirochætes. Balfour still claims to have produced the disease by means of the granules. Marchoux and Couvy, however, failed to do so and consider Balfour's results as due to thin spirochætes which were undetected. The writers have often observed thick and thin organisms, but in examinations of over 5,000 blood smears, including some from natural cases, have failed to see any granulated red cells.

Montgomery (1908) and Balfour (1911) are of opinion that the disease increases in severity in passage from bird to bird. Fulleborn (1909) has conserved the Brazil strain of spirochætes for seven years in canaries, the virulence having been maintained throughout. Marchoux (1907) and Anderson (1928), however, found that on passage of *Sp. gallinarum* from fowl to fowl the organism loses its pathogenicity but that its virulence may be maintained by passage through an intermediary host. Blaizot (1909, 1910) found that in chickens five to twenty-one days old, inoculated with infected blood, the course of the disease is longer in the earlier passages than in the later ones, in which also no tangle formation was observed before death. He concludes by saying that the Somaliland and

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Brazil strains become attenuated by passage through adult birds but that passage through chickens enhances their virulence.

Contradictory statements, such as those quoted, suggest that the parasite may behave differently in different environments. It is obviously necessary, therefore, to study the farms concerned in efforts at control.

The geographical occurrence of spirochætosis in India is not definitely known. Preliminary reports from the Punjab and Central Provinces confirmed the occurrence of the disease in those parts, and in recent years it has been reported from other parts of India as well. In Mukteswar, *A. persicus*, the transmitter of the disease, abounds in fowl-pens where healthy birds intended for experimentation are kept.

Maintenance of the Strain

At first, the strain of spirochætes was maintained in fowls by direct passage, but, later, blood containing spirochætes was stored in a refrigerator with sodium citrate and passaged through fowls once in three weeks.

Leadingham (1935-36) reports that one drop of 2 per cent. sodium citrate added to a cover-slip preparation from a lesion of Vincent's angina caused immediate cessation of movement and eventual dissolution of the spirochætes. He even treated ulcers in a patient successfully with this drug. However, even with four parts of blood and one part 4 per cent. sodium citrate, the writers have not noticed any spirochæticidal action, at least, against *Sp. gallinarum*. Altogether, up to the present time, the strain has been through twenty-four passages in forty-six fowls during sixteen months; of the forty-six fowls, thirty-four recovered without treatment and only six died. This suggested that the strain had undergone attenuation but it was uncertain whether such attenuation was due to refrigeration or to bird passage. After thirteen passages the strain was tested on a number of healthy birds and, although they were affected, they did not succumb. At this stage the strain was again passaged direct from fowl to fowl. After five direct passages the birds began to show acute symptoms and many of them died of the disease. It therefore appears that refrigeration was the attenuating factor and that subsequent direct passage again exalted it.

By refrigeration for about two weeks the parasites are sufficiently attenuated to be used as a vaccine under certain conditions, although recovery of all vaccinated birds cannot be guaranteed. Under refrigeration, the parasites remained alive for at least twenty days, often for fifty to seventy days, and in one instance up to ninety-seven days. In all cases, where moving organisms could be found, the sample proved to be infective. Blood samples, in which the parasites were motionless, were found to give some immunity, although the samples were not infective.

Course of the Disease

Infection.—It has already been established that the disease is transmitted from fowl to fowl by *A. persicus* in all its stages. The mechanism of transmission had been fully dealt with by Knowles *et al.* (1932). The writers, however, found that adult ticks do not transmit the disease as readily as larvæ. Dodd (1910) failed to produce the disease with adult ticks but with nymphs and larvæ

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infection was always transmitted. Gilruth (1910) recorded a case where only the larval forms produced the disease. Bevan (1908) kept about thirty birds in a stand which had been left unoccupied for six months. On the second day all the birds were smothered with larval ticks and on the next day all showed symptoms of the disease. Williamson (1908) observed that in Cyprus the disease is common in summer and absent in winter, while it is more common in runs where the conditions are suitable for the hatching of tick eggs. Similar observations have been recorded at Mukteswar. Birds kept in a tick-infected run develop the disease during summer but seldom in winter; on examination only larval ticks are found on the birds. This suggests that it is the larvæ of *A. persicus* which are the real vectors.

In the absence of the vector, infection can also take place by cohabitation of healthy birds with sick ones. Two healthy birds were kept with a sick one in a tick-free cage and both developed the disease.

Incubation period.—From the time of inoculation of virulent blood to the rise of temperature and the appearance of the spirochætes in the circulation the incubation period varied from one to five days, averaging one to two days. In the case of natural infection the period of incubation was three to eight days. Knowles *et al.* (1932) have pointed out that the incubation period depends upon the quantity of infective material used and the route of infection, the intramuscular route being the most rapid and certain method after the intravenous one. For purposes of comparison, the writers injected 0.2 c.c. passage blood diluted with an equal amount of citrate solution into four groups of five birds as follows: (*a*) intravenously, (*b*) subcutaneously, (*c*) intramuscularly and (*d*) by smearing on scarifications on the comb. Though the temperature reaction was observed to occur a little earlier in groups (*a*) and (*b*), the spirochætes appeared in the circulating blood almost simultaneously in the first three and only a day or two later in the fourth batch. Altogether about 2,000 birds have been infected during the course of this work both by intramuscular and subcutaneous routes and no difference in the incubation period has been noted, always, of course, provided the material and quantity used is uniform.

Symptoms.—Although it is not possible to make a sure diagnosis without the aid of a microscope, certain symptoms may raise a suspicion of spirochætosis.

In 500 healthy birds the normal temperature was found to vary from 106 deg. Fahr. to 107.5 deg. Fahr. The disease starts with a rise to 108 deg. Fahr. then to 110 deg. Fahr. or beyond. At the first rise of temperature blood smears may fail to show parasites, but they appear within the next twelve to twenty-four hours. When parasites are numerous the temperature may fall and finally become subnormal before death. Kapur (1940) stated that in fowls infected from ticks there was no appreciable rise of temperature synchronising with the appearance of spirochætes in blood. Out of sixteen fowls observed, only five showed a rise of temperature. However, in the present observations on more than 2,000 birds, infected either artificially or naturally, very few positive cases failed to show an appreciable rise of temperature at the onset. The temperature charts representing both experimental and natural disease, are those of some typical cases. (Table I.)

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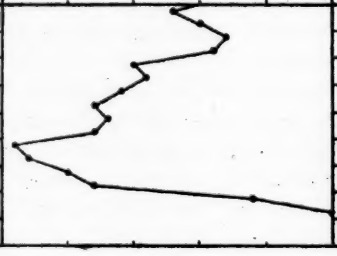
TABLE I

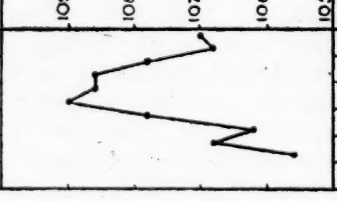
TEMPERATURE CHARTS

NATURAL ATTACK BY TICK INFECTION



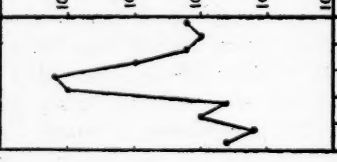
Symptoms & Treatment	Smear Examination
	Negative.
	〃
Dull & sitting in a corner	〃 Eosinophilia
〃 〃 thirsty	Spirochaetes +
Characteristic posture	〃 ++
Sleepy	〃 +++ Monocytosis
Off feed	Tangle formation
Moribund stage	Negative
Died.	

EXPERIMENTAL CASE - ROUTINE PASSAGE



Symptoms & Treatment	Smear Examination
Dull	Negative
〃	〃 Eosinophilia
〃	Spirochaetes +
〃	〃 ++
〃	Numerous
〃 Recovered.	Negative.

EXPERIMENTAL CASE SULFARSENOL TREATED



Symptoms & Treatment	Smear Examination
	Negative
Dull	〃
〃 Sulfarsenol 1/2 gr.	Spirochaetes +
Recovered.	Negative. Lymphocytosis
	〃

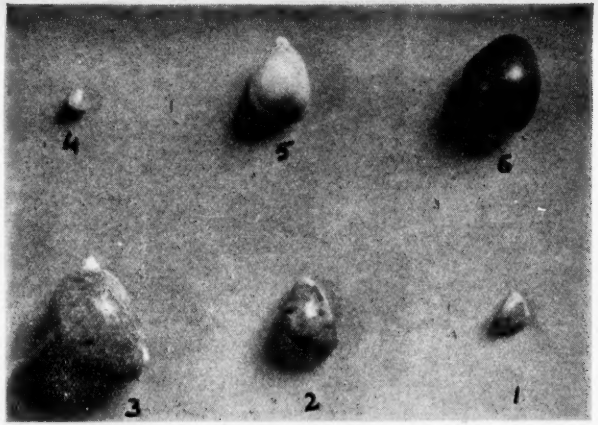


FIG. 1.

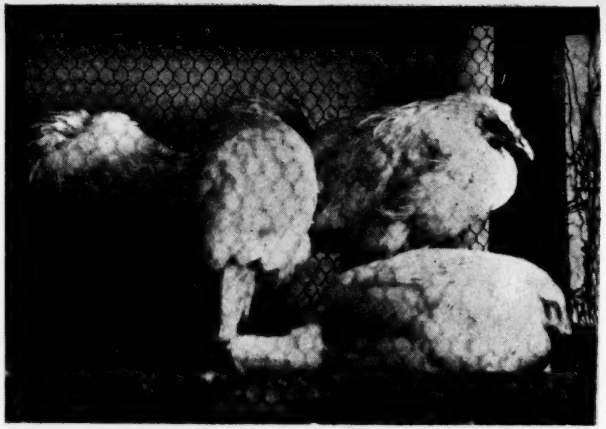


FIG. 2.

PLATE I.

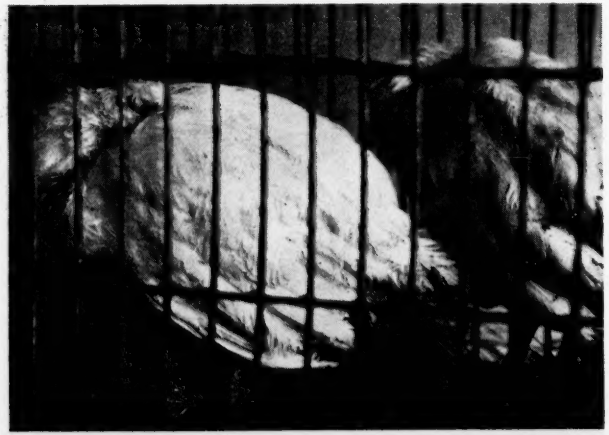


FIG. 3.

FIG. 1.—Spleens from healthy and infected birds. No. 1, Healthy adult spleen. Nos. 2, 3, 5 and 6 are from adult birds died of spirochætosis. No. 4 is the spleen from a 3-week old chicken dead of spirochætosis (apparently normal).

FIG. 2.—Birds at different stages of the disease.

FIG. 3.—Early stage of spirochætosis—birds in characteristic posture.

PLATE II.

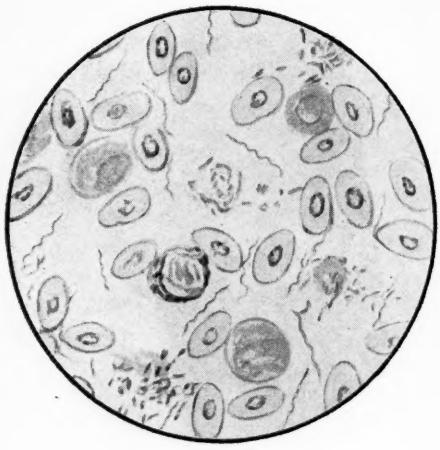


FIG. 1.

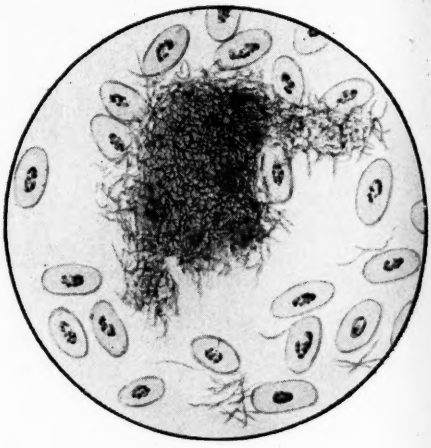


FIG. 2.

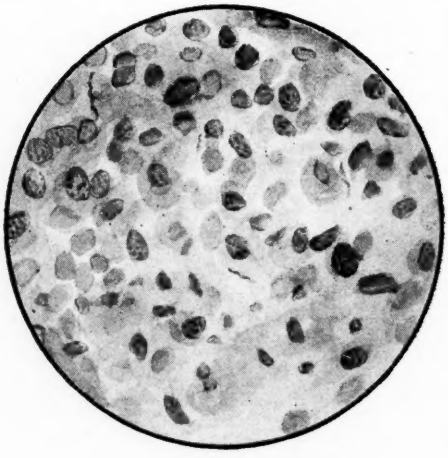


FIG. 3.

FIG. 1.—Stained blood smear from a bird suffering from spirochætosis. Note the pseudoeosinophiles in different stages with granules and also the monocytes.

FIG. 2.—Tangle formation at crisis.

FIG. 3.—Section of spleen showing spirochætes at the height of infection (Levaditis method).

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With the rise of temperature some birds show slight dullness, while others remain active. When parasites appear in the blood the birds become dull and assume a posture which, although somewhat characteristic, is not diagnostic, viz., with the head buried under the feathers of the back. (Plate I, Fig. 1.) Rise of temperature as a rule is followed by intense thirst. As the disease progresses, profuse diarrhœa, ruffling of the feathers, loss of appetite, a crouching position, dullness, disinclination to move and rapid emaciation are noticed. In the last stage the head droops down and the eyes are closed. (Plate I, Fig. 2.) In the terminal stage the symptoms are again identical with those of other acute diseases of poultry.

Mortality.—In natural outbreaks this is high. In one outbreak among twenty-five chickens in a tick-infested cage twenty-one deaths occurred from acute spirochætosis. Owing to the intensity of the infection, the onset was rapid and treatment could not be applied in time. Mortality varies with age, breed, condition and certain other causes. Birds of improved breeds such as the Rhode Island Reds and White Leghorns show acute symptoms and, even when in good condition and above one year old, seldom recover, even from experimental inoculation, unless treatment is commenced as soon as the parasites are noticed in the circulating blood.

Of the predisposing causes the most important one observed so far is visceral gout, a condition mostly seen in the Rhode Island Reds. It is said that visceral gout is caused by ill-balanced rations or the feeding of excessive proteins. Whatever may be the cause of death, more than 60 per cent. of the adult birds that died as a result of the present experiments showed extensive visceral gout with whitish deposits on the heart, liver, kidneys and spleen. The mortality is least in birds between six and twelve months of age, and such birds usually recover easily with chemotherapy.

After-phase.—Of the 2,000 birds dealt with in the present enquiry, 98 per cent. were uncomplicated cases. Among the few complications met with, by far the most important was paralysis of the wings or legs, or both. This paralysis may have been caused through capillary embolism by clumped organisms, since with the disappearance of the clumps, symptoms passed off in most cases. The only treatment required was massage of the part with a stimulating liniment.

Blood picture.—In smears stained by Leishman or Giemsa methods the number of spirals in the parasites varies from five to eighteen. Single isolated organisms which are seen earlier are generally longer than those met with later. Together with the parasites there are seen under dark ground illumination small actively moving coccoid bodies which are absent in healthy blood preparations. Whether they are stages in the development of the spirochætes or the so-called "Balfour's granules" is uncertain. As soon as one or two organisms per field are seen, treatment should be commenced. On the fifth or sixth day, the parasites have multiplied to such an extent that they are more numerous than the corpuscles. This is followed by tangle formation which marks the crisis of the disease. Two or three tangled masses may be seen in each field and each mass may be formed of hundreds or even thousands of parasites. (Plate II, Fig. 2). After the crisis, the bird either recovers or dies. In the former case the parasites disappear from the blood.

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Apart from the anæmia caused by the disease, the most important hæmatological change is leucocytosis. In the healthy fowl the mean white cell count is 30,000 to 45,000 (Launoy and Lévy Bruhl, 1914). Soon after infection there is a sudden wave of leucocytosis, chiefly of polynuclear type (pseudo-eosinophiles of Launoy and Lévy Bruhl). The appearance of these cells indicates infection, though this is not entirely diagnostic.

The granules of the eosinophiles, seen at first as pink bodies, increase in size and finally appear as gram-negative, rod-shaped bodies before they are set free in the blood stream. (Plate II, Fig. 1.) This stage is followed by a decrease in the number of eosinophiles and an increase in the mononuclear cells. In certain monocytes, groups of pink, round granules are seen in the cytoplasm. These also disappear after a day or two leaving behind groups of vacuoles. Similar pink granules have also been observed sometimes in blood smears. These are extra-cellular and are embedded in an irregular cytoplasmic mass. In a recovering bird, in the place of monocytes, small lymphocytes appear in large numbers.

Morbid Anatomy

Lesions are almost entirely restricted to the liver and spleen. The spleen may be enlarged to about six times the normal. (Plate I, Fig. 3.) The liver is pale, shows evenly spaced, yellowish, necrotic areas and in severe cases is more friable than usual. There is also biliary stasis. The heart muscle is congested and petechiated. If death takes place in the after-phase, splenic enlargement is not constant. The same is true of cases treated with an effective drug. In any case, whether treated or not, parasites disappear from the blood after death. Smears taken from the spleen, liver and heart blood about three hours after death failed to reveal spirochætes, though sections prepared from the spleen, liver and brain from a bird destroyed at the height of the infection revealed these parasites. (Plate II, Fig. 3.)

Summary

A strain of spirochætes was maintained by three-weekly passage of blood, stored in the intervals in a refrigerator. Spirochætes stored in refrigerated blood may remain alive up to seventy days or more. The organisms in refrigerated blood are attenuated but become exalted on subsequent direct passage through fowls.

In respect of transmission of the disease, the larval form of *A. persicus* is more infective than the adults. Infection is also possible by cohabitation.

The period of incubation is dependent upon the concentration of the infective material rather than upon the route of inoculation. There is invariably a rise of temperature before the parasites appear in the blood.

Mortality in natural outbreaks was 60 per cent. or even more. Rhode Island Red fowls were found to be more susceptible, the death-rate among this breed being also very high. The most common predisposing causes of mortality among adult birds are visceral gout, high breeding and obesity.

In some recovered birds, paralysis of the wings and legs was observed.

Enlargement of the spleen is a common post-morten feature.

(*To be continued*)

DISTEMPER IN DOGS

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Literature

NICOLLE[1] (1931) carried out an experiment in which he inoculated 5 c.c. of the virus of distemper into puppies. They all developed the usual symptoms. Five c.c. of blood from these puppies were given to each of a man, a young monkey and a puppy. The puppy developed the characteristic curve of temperature on the fourth day and passage of blood from this animal established the presence of the disease. Neither the man nor the donkey gave any evidence of infection, febrile or otherwise. On the other hand, a dog which received blood from the man taken on the sixth day after inoculation gave a characteristic temperature curve and the disease was passed to the other dogs.

From this experiment the author concludes that man may be a carrier of the disease.

Laidlaw and Dunkin[2] (1932) say that the virus content of the liver is very variable and may be sometimes quite small, and thus they do not recommend the use of this organ for vaccine production as a routine.

Laidlaw and Dunkin[3] (1933), after ten years' research, found the cause of primary distemper was proved to be an ultramicroscopic filter-passing virus. It can be readily transmitted from dog to ferret and vice versa.

A healthy dog can be given lasting protection against the disease by the inoculation of vaccine followed by an inoculation of virus a fortnight later. Later it was found that an animal could be immunised by the simultaneous inoculation with antiserum and virus. Antiserum alone is of value in the early stages of distemper, as it lessens the severity of the attack.

Thomsen[4] (1933) gave the results of testing the protective power of Laidlaw-Dunkin method (vaccine plus virus) against distemper. Out of 876 dogs vaccinated in Denmark between August, 1931 and 1932, only fourteen dogs contracted distemper after both injections had been given, two dogs had distemper when the immunisation was commenced, five were attacked within seventy-two hours after the first injection and fourteen before the second injection.

Failure of this method of immunisation thus occurred in four to five per cent. of the cases only.

The author considers this method as highly advisable.

Slagsvold[5] (1933) found that vaccination with Laidlaw-Dunkin vaccine gave satisfactory results, only two dogs out of 180 vaccinated developed distemper a few months after vaccination.

Dogs evidently carry infection for some weeks after vaccination, as susceptible puppies placed in contact became infected. Dogs, therefore, ought to be isolated for about one month after vaccination.

Little[6] (1934) says that the simultaneous injection of serum and virus produces a strong lasting immunity. Evidence is given that a more powerful immunity is produced if forty-eight to seventy-two hours are allowed to pass after injecting the virus before administering the appropriate dose of serum. The author claims perfect safety in immunising a young dog by the serum-virus method.

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Lehnert[7] (1934) gave details of an experiment with killed virus in a ten per cent. brain-suspension sterilised by boiling. From the results obtained he drew the conclusion that one or two successive subcutaneous inoculations (given at three-day intervals) of 5 c.c. of suspension containing killed virus set up a condition in dogs in which there is resistance to a subsequent inoculation of living distemper virus, carried out three to twenty-eight days after vaccination.

Slanetz[8] (1935). From his experiments the author concludes that the intradermal inoculation of young puppies with living distemper virus within a month of birth does not give rise to distemper. He also states that, despite the absence of disease symptoms, the injection appears to confer a permanent immunity.

Plantureux[9] (1935). The author describes the results obtained with anti-distemper serum prepared from dogs, already naturally resistant to distemper, by enhancing the immunity with one inoculation of 20 c.c. of formalised virus. The dogs were bled to death ten to twelve days after receiving the formalised virus. The sera of thirty to fifty dogs were mixed but no attempt was made to concentrate the serum. The dose of the serum was computed on the basis of 3 c.c. to 6 c.c. per kilogram body weight.

If the serum was administered on or before the third or fourth day of illness, before the invasion of secondary organisms, very satisfactory results were obtained. If the response was not entirely satisfactory, or if the serum was given later, it was found of benefit to give a second or even a third dose of serum.

Goret[10] (1936) treated twenty-seven cases with the homologous anti-distemper serum made by the method described by Plantureux (see above). The author considers that, if employed before the appearance of bronchopneumonic complications, the serum is an effective therapeutic aid. He points out, however, that though in his experience one dose of 20 c.c. of serum may cause an apparent recovery, a second dose should be given from eight to fifteen days to prevent a relapse. Even when complications of distemper have appeared, the serum should be administered, as, in these cases also, it is definitely of value.

Green [11] (1940) used a distemper virus which had been passed through ferrets fifty-three times for the immunisation of 419 dogs between twelve weeks and five years of age. A single subcutaneous dose was given in all cases. Only four died; all the others resisted artificial and natural infection.

Whitney [12] (1940) considers *the commercial viruses to be useless because they are usually dead.* Whitney considers that it is possible to confer a solid immunity on dogs by the use of sufficiently large doses of a formalised vaccine which has been freshly prepared from the spleen of an animal at the height of infection.

History and Technique

At a dog's training station near Cairo, the following cases were studied:

Case 1.—An Alsatian cross, eighteen months old, indisposed on October 16, 1942, died on October 31, 1942, running a temperature most of the time varying between 103 to 105 deg. Fahr., commencing with loss of appetite, laryngitis, salivation, a little nasal discharge, slight anæmia, stool fairly normal, no fits and no paralysis, spirit kept up all the time, blood films negative to piroplasmoses.

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The dog was given M & B tablets on four successive days and tryparsamide S/C with no effect. *P.M.* picture not available.

Case 2.—Eight months Alsatian reported ill on October 26, 1942, and died on October 30, 1942. Appetite depraved, temperature almost normal, condition wasted rapidly, diarrhœa, fæces tinged with blood, slight nasal discharge, slight bleeding from gums. *P.M.:* Pneumo-enteritis. Blood films negative.

Case 3.—Two years old cross Alsatian, took ill on October 28, 1942, died on November 4, 1942. Slight rise of temperature, off food, tender over kidney area, breath bad, absence of paralysis and fits. *P.M.:* Pneumonia. Blood films negative.

Case 4.—Eight months old Alsatian, indisposed on October 27, 1942. Temperature 103 deg. Fahr., off food, slight loss of condition, slight nasal discharge, no abdominal symptoms. On November 18, 1942, the dog developed fits of repeated but intermittent nature, until death on November 20, 1942. *P.M.:* This is the first post-mortem I personally performed. Congested lungs, gastro-enteritis, liver swollen, gall bladder distended full of bile, spleen slightly enlarged.

Blood films, blood swabs, pieces of lungs, intestines and the whole liver as well as the spleen were aseptically collected, films were negative to protozoa and micro-organisms, cultures ærobically and anærobically incubated were also negative.

Two rabbits, two guinea-pigs and two mice were intramuscularly inoculated with ½ c.c. of blood, each with negative result.

Having been convinced of absence of micro-organisms and protozoa, and being anxious to save the other dogs not awaiting confirmatory diagnosis, equal parts of spleen and liver were minced and emulsified in 50 per cent. glycerine saline, 2 gm. by weight of tissue being added to every 100 c.c. glycerine saline, commercial formalin was added to make up 1/500 dilution. This antigen was shaken daily and kept in an ice-chest three days before use and the following dogs were injected with five successive doses, 5 c.c. a dose, intramuscularly every three days.

Case 5.—Cross Alsatian commenced indisposition on October 26, 1942, this dog's main symptoms being nasal discharge, severe diarrhœa, passing almost pure blood, intense eye discharge, eyes almost completely closed. On November 25, 1942, 5 c.c. vaccine given, severe constitutional reaction, the dog looked almost prostrated, but eight hours later the dog revived and completely recovered on December 5, 1942. The dog, however, received his five injections and was discharged on January 11, 1943.

Case 6.—Eighteen months Alsatian, took ill on October 28, 1942, showing a rise of temperature 103 deg. Fahr., loss of condition, bad breath, nasal discharge, obstinate diarrhœa. The dog was kept alive through splendid nursing. On November 25, 1942, vaccine was given, very severe reaction, eight hours later almost prostrated, but by midday November 26, 1942, temperature had dropped to normal and the dog looked brighter. A full course of five injections was given. The dog recovered and was discharged on January 11, 1943. It gained 6 pounds in weight before being discharged.

Case 7.—One-year-old Alsatian on October 28, 1942, developed a tempera-

ture. The temperature dropped slightly on injecting 2½ c.c. streptocide "Evans" but appetite did not improve. A few days later temperature went up again. Starting on November 25, 1942, a course of vaccine was administered. The animal recovered and was discharged on January 11, 1943.

Cases 8, 9 *and* 10.—All Alsatians, main symptoms were rise of temperature, appetite depraved, condition lost. They were treated with vaccine and recovered.

Experimental

The previous ten cases encouraged one to carry on, but as no ferrets or weasels were available, stray dogs at Abbassia Isolation Hospital were used with the kind permission and every support of Dr. I. F. Salem Bey, Director of Veterinary Service, Egypt.

All the dogs used were mongrels; young ones were selected over six months old and under two years. Ten unweaned puppies were inoculated with ½ c.c. blood each from Case No. 4, and there was no reaction. Of seven other dogs between six and eighteen months old, two reacted. There was a distinct rise of temperature to 104 deg. Fahr. twenty-four hours after the injection of ½ c.c. blood from Case 4. There was nasal and eye discharge seventy-two hours following the inoculation.

Blood of reactors was collected and frozen in order that repeated injections might be made every three weeks to keep the virus going. The spleen and liver of reactors was also preserved in 50 per cent. glycerine saline for vaccine production.

Some of the non-reactors were bled to death under chloroform for immune serum production.

Vaccine Preparation

The two per cent. spleen liver emulsion 1/500 formalin having proved helpful, a ten per cent. emulsion was substituted and formalin 1/1,000 was added five days before use; the five doses were substituted for two doses of 5 c.c. and 10 c.c., the latter given after a week's interval.

This vaccine has been used as both prophylactic and curative in about 200 cases with satisfactory results. Serum has not yet been used.

Mongrel dogs, through infection and recovery, or hereditary resistance, develop a reaction only in about 30 per cent. of cases inoculated with virulent blood.

Summary

(1) Uncomplicated distemper reveals itself in a rise of temperature and depraved appetite as well as by inflammation and inflammatory discharge of mucous membranes of the eyes and nasal chambers.

(2) Ten per cent. emulsion of spleen and liver in 50 per cent. glycerine saline 1/1,000 formalin is both safe and encouraging in treatment and prophylaxis. There is little or no reaction, and absorption is complete in a few hours.

(3) When ferrets and weasels are available, further experiments will be reported.

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PIGEON POX (MAY, 1943)

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Literature

JAMES[1] (1931) states that the mouth form of fowl pox is the most prevalent type met with in Illinois and that the mortality is low. He carried out immunity experiments, using a vaccine consisting of a saline suspension of powdered pox crusts.

Inoculations were made either by brushing the vaccine on to a plucked area of skin or by stabbing the skin with a sharp-pointed scalpel which had been dipped in vaccine. Both of these methods confer a solid immunity.

Doyle[2] (1932). Experience has shown that vaccines prepared from unmodified fowl pox virus confer a lasting immunity, but are unreliable and dangerous in action. There is usually loss of condition in the treated fowls and, frequently, there is generalisation of infection while occasionally heavy mortality is encountered.

Vaccine prepared from pigeon pox is used exclusively in this country and, although the immunity conferred by it is less than that given by vaccines containing fowl pox virus, it is sufficient to protect against natural infection and has the additional value of being free from danger.

Glover's[3] (1931) experiments with unattenuated fowl pox virus showed that this method was followed by fairly extensive local lesions with scab formation and that there was sometimes a considerable loss in condition.

He found that heated virus conferred a short immunity which was weakened by the eighth week and had entirely disappeared by the twenty-fourth.

Vaccines attenuated by phenol, formalin and chloroform had a little value and conferred immunity for a short time only.

Pigeon pox virus was found very effective in protecting fowls against severe natural infections of fowl pox.

Kligler, Komarov and Fiat[4] (1933) found that the pigeon pox vaccine produced mild, rapidly-healing lesions in fowls while the living fowl pox vaccine caused systemic disturbance and in a greater or lesser percentage of fowls generalisation of the infection occurred.

Irons[5] (1934) found that in chickens inoculated with pigeon virus the lesions remained localised and recovery was complete at the end of three weeks. The chickens proved on subsequent test to be protected against fowl pox virus.

Attempts to adapt bird pox virus to mammals gave negative results.

Matsumura[6] (1934). The author adapted fowl pox virus (Japanese strain) to *rabbits by testicular passage.* The virus was adapted after the fourth passage and was then capable of producing a pustular eruption on the skin of rabbits.

Similar experiments were carried out with fowl pox virus of American and of Korean origin, but neither was so readily adapted to the rabbit as the Japanese strain.

Doyle[7] (1934) states that the most satisfactory method of preparing the pigeon pox virus vaccine to protect fowls against fowl pox is to remove the crusts from the inoculated areas on the pigeon's breast, dry rapidly over P_2O_5 *in vacuo* and store in sealed tubes at 2 deg. Cent. Immediately before use sufficient crusts are powdered and suspended in glycerine-saline to make a one per cent. suspension.

Doyle[8] (1935) considers the inclusion bodies present in the cells in the skin lesions to be the causal agents. Suspensions of the elementary bodies contained in the cell inclusions are agglutinated by sera from recovered birds.

Certain biting flies and mosquitoes are capable of transmitting fowl pox, but direct infection from diseased to normal fowls is probably the most important method of transmission.

The unattenuated pigeon pox virus is the best method of immunisation. It does not cause constitutional disturbance nor risk of generalisation of infection.

It is recommended that the annual inoculation of all young stock with pigeon pox virus should be a routine procedure.

Beaudette and Hudson[9] (1938) described a method of obtaining a pure pigeon pox virus in culture on the chorio-allantoic membrane of embryonated eggs, nine to fifteen days old, followed by serial passage through nineteen generations of tissue culture. On account of the great affinity this virus has for follicular cells, which are always subject to bacterial contamination, Berkefeld filtration was essential for the primary culture. Most growth occurred after five days' incubation of the inoculated eggs, the lesions produced in the membranes being identical with those due to fowl pox. The virus withstood up to thirty-eight days' refrigeration between passages, but the virulence of infected membranes was slightly reduced thereby. In neutralisation tests with virus and immune serum there was an absence of marked generalised infection of inoculated membranes as compared with lesions produced by virus alone.

Membranes dried *in vacuo* gave " takes " when chickens were inoculated by scarifications with concentrations of 0.625 mg. (or more) per c.c. of 50 per cent. glycerine solution.

Brandly and Dunlap[10] (1939) passed the viruses of fowl pox and pigeon pox serially through eggs. A strain of fowl pox virus retained its normal virulence and pathogenicity after sixty-eight successive serial egg passages at irregular intervals, including the use of eggs of other species of birds (ducks, guinea-fowls, turkey). The pigeon strain was carried through fifteen egg

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passages. The authors obtained growth of the virus in tissue culture, although not so abundantly as in eggs.

The egg-passaged (bacteria-free) virus was found to be superior to skin-lesion (bacteria containing) virus as an immunising agent of chickens against fowl pox.

Coronel[11] (1939) used a glycerine-saline emulsion of fowl pox virus as a vaccine. No difference was discernible in the immunising value of the vaccine with varying concentrations of virus.

The efficacy of the different methods of vaccination was investigated and the author found that the stick method, inoculating into the flank, was the best. Attention is drawn to the danger of vaccinating chicks under one month old, especially day-old chicks. When pigeon pox virus is advocated as a vaccine against fowl pox in chickens, the author recommends double vaccination, i.e., inoculation with pigeon pox virus first, followed by fowl pox virus; this was found to reinforce the diminishing immunity in birds about three months after vaccination with pigeon pox virus.

History

Twenty-two pigeons were received at the school laboratory on March 4, 1943, from a pigeon breeding station near Cairo. Twenty birds arrived dead (destroyed at the point of death) and two birds were alive. All the birds were homers out of a breeding centre containing some few hundreds of homers, all British-bred birds.

No distinct lesions were seen on skin or mucous membrane of the destroyed birds, but the two living adult birds, a blue hen and a red cock, showed typical clinical pox lesions, mainly on mucous membrane of the mouth and few pox nodules round the eyes and the nasal orifices.

Experimental

Experiment 1.—Post-mortem was made on all the destroyed birds but no outstanding lesions were seen on any.

Blood films and blood cultures incubated ærobically and anærobically were *negative.*

Three rabbits and three Egyptian bred young pigeons were intramuscularly inoculated with the mixed blood of the destroyed pigeons with negative result.

Experiment 2.—The skin and mucous membrane lesions of the two living birds were emulsified in a mortar in 80 per cent. glycerine-saline and left in ice-chest for three days before use to destroy accidental bacteria proved by film and culture.

The emulsion was one per cent. (1 grm. lesion in 100 c.c. glycerine-saline); 20 c.c. of this was passed through Sietz filter 2.5 inches in diameter, 70 mm. pressure at 22 deg. Cent. room temperature for ten minutes; the filtrate was scarified over the upper mucous membrane of the palate of two pigeons. Three other pigeons were scarified with the non-filtered emulsion. All the six birds developed typical pox lesions by the fifth day. *This proves the disease to be an outbreak of small pox.*

N.B.—All experimental pigeons or fowls used were Egyptian bred and young, two to five months old, unless otherwise mentioned.

Experiment 3.—Three young fowls purchased from two different dealers

were scarified with the non-filtered emulsion used in Experiment 2. The birds were kept under observation for fifteen days, two did not develop any lesion whatever at the seat of scarification of the palate while the third developed a very small elongated lesion about 5 mm. long and 1 mm. wide on the third day of scarification which disappeared altogether by the seventh day.

The pigeon virus does not pass in fowl to produce a crop for vaccine production.

Experiment 4.—Two rabbits were intradermally inoculated on their shaved backs, one with emulsion of pigeon pox lesion in 80 per cent. glycerine after *immediate emulsion* (which on microscopic examination contained various accidental micro-organisms) and the second with the same emulsion after being three days in glycerine-saline (films were free from micro-organisms). The latter was kept under observation for a fortnight and no lesions were seen, but the former developed blisters after three days, scabs on the seventh day. The blister contents were scarified on the palate and thighs of two pigeons with negative results and the scab emulsion collected from the rabbits and treated with 80 per cent. glycerine in a thick emulsion, about 3 per cent., after being left for three days to destroy accidental bacteria, was intradermally inoculated into a third rabbit with negative results. *False lesions* simulating pox may be produced by accidental bacteria on intradermal inoculation into rabbits and apparently the virus is not transmissible to mammals.

Experiment 5.—Five pigeons were intramuscularly inoculated with pigeon virus emulsion, 1 per cent., each receiving 0.5 c.c. (one pigeon control was scarified on the palate with the same emulsion). The birds were kept under observation for fifteen days without showing any pox lesion, while the control developed lesions within the fifth day of the scarification. The five birds were then scarified at the palate with control 1 per cent. pigeon pox virus emulsion. Only one pigeon developed lesions at the seat of test.

These experiments indicate that the most successful route of infection is scarification.

Vaccine Production

Vaccine No. 1

Experiment 6.—A 0.5 per cent. pigeon scab emulsion plus 5 per cent. liver and spleen emulsion of an infected pox pigeon in 70 per cent. glycerine to which formalin was added (1-500) was prepared. It was kept in an ice-chest for three days before use. Three homers were scarified on the right thigh after a few feathers were pulled out. All reacted and scab, flat, brownish, non-extensive, developed by the fifth day and dropped off by the tenth day. Three weeks later virulent controlled 1 per cent. pigeon pox virus was scarified on the left thigh of the same birds without any reaction.

Formalin in this case did not interfere with the protective properties of the virus.

Vaccine No. 2

Experiment 7.—Recovered pigeons were bled to death and serum collected. To each 1 c.c. serum two drops of 1 per cent. pigeon pox virus, 80 per cent. glycerine, were added and incubated for two hours. Five pigeons were scarified with this preparation but all developed severe pox lesion within the seventh day

of scarification. Lack of funds did not allow further investigation of this point.

Vaccine No. 3

Experiment 8.—Fowl pox lesions, 2 gms. by weight (collected from pox naturally infected fowls), were added to pigeon pox lesion 1.5 gms. by weight. These were put in a small petri-dish in an anærobic glass jar (Flides and MacIntosh jar), in presence of caustic soda sticks and pure sulphuric acid for five days. On being weighed again 0.5 gm. was lost. An emulsion, 1½ per cent., was made in 80 per cent. glycerine-saline. Six homers were scarified on the left thigh after a few feathers had been pulled out. All reacted by the fourth day of vaccination, the scab healing by the tenth day. Two weeks later these birds were scarified on the right thigh with controlled 1 per cent. pigeon pox virus and all proved immune. This vaccine was used on 6/8 homers (up to now) which all reacted except two birds. The reaction was mild, a small scab formed by the fifth day of vaccination without interfering with the general condition of the birds or their feeding capacity.

Experiment 9.—Four infected pigeons—lesions over palate and round eyes were touched daily with concentrated watery solution of potassium permanganate without curretting lesions—the four birds recovered in ten days. Great care was taken that no permanganate solution reached the throat.

Experiment 10.—The virus (Seitz filterate, 5 c.c. 1 per cent. emulsion, also 5 c.c. non-filtered 80 per cent. glycerine-saline emulsion, as well as 5 c.c. blood of an infected pigeon at the early stages) were separately seeded on solidified pigeon blood, also pigeon serum agar tubes incubated ærobically and anærobically at 39 deg. Cent. for ten days with negative result.

Summary

(1) The pigeon pox virus does not pass through fowls.

(2) The pigeon pox virus, after being freed from contaminant micro-organisms does not produce false or true lesions in rabbits.

(3) Both dried and formalised emulsion are protective.

(4) Potassium permanganate gives good results in treatment.

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XUM

Clinical Cases

RUPTURED VAGINA IN THE HEIFER

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Subject.—White shorthorn heifer.

History.—Had been in labour eight to ten hours, but straining had not been excessively violent.

Examination.—The heifer was recumbent when first seen but could rise with ease. She appeared well in herself and did not strain excessively. Vaginal examination revealed the presence of a large body between the vagina and the rectum which was forced backwards within a few inches of the upper commissure of the vulva at each strain on the part of the heifer. Further examination revealed that this mass was the head and forelegs of the calf in the anterior normal presentation. It felt as though the fœtus had passed into another passage at the cervical end of the vagina and was situated above the vagina and below the rectum. The end of the intervening septum could be felt at the cervical end. This septum was, in actual fact, the torn roof of the vagina. No hæmorrhage was noticed at all and no blood was found on the arm after examination.

Operation.—Epidural anæsthesia was induced using 13 c.c. of Parsetic. Tail relaxation was obtained almost immediately and all straining ceased after ten minutes. The heifer remained standing. It was found possible to get one's hand around the cervical end of the septum (torn vaginal roof) and in turn grip the cannon bone of each foreleg, reducing each back into the vaginal passage. Lastly the head was manipulated into the vagina. This procedure was found to be quite easy under epidural anæsthesia. It would have been entirely prevented by the abdominal straining in its absence.

The calf was delivered alive but died almost immediately. The vaginal passage was small and difficulty was found in delivery. No hæmorrhage was observed throughout. After delivery, a vaginal examination revealed a large rent in its roof at the junction of the cervix and the vagina. Blood was noticed on the arm when withdrawn this time.

A grave prognosis was given and the heifer died three days later.

Comment.—It is interesting to note that the calf was presented normally: the cervix was widely dilated, there was no abnormal straining and there had not been any previous lay interference. No abnormality could be discovered in the genital tract to account for the production of the lesion.

AN UNUSUAL VAGINAL FIBROID IN THE BITCH

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Subject.—Alsatian bitch; six to seven years old; maiden.

History.—Œstrus six months previously, slight discharge from the vulva. Difficulty in urination and defæcation for three days. It was because of these latter symptoms that advice was sought. No history of any illness.

Examination.—Abdominal palpation found a large growth in the pelvic region

about the size, shape and position of a distended bladder. Vaginal examination was impossible owing to its smallness in size. Under Nembutal, the bladder was found to be grossly distended and the growth to be situated above the bladder and extending back into the pelvis. There was no rectal impaction.

Diagnosis.—Growth in the uterine body.

Treatment.—It was decided to perform a flank laporotomy to discern the exact nature of the lesion and remove it if possible. Nembutal anæsthesia was employed and the usual technique of a flank incision followed. The uterine cornua were found to be normal but a large, hard, elongated mass was seen and felt in the region of the uterine body and anterior vagina. It was hanging over the pelvic brim, dragging the uterus and anterior vagina with it, and at the same time projected back into the pelvis. Owing to its size, it was found impossible to get the mass near the incision and as it seemed impossible to remove it satisfactorily, it was decided to destroy the patient.

Post-mortem.—A large fibroid attached to the ventral wall of the anterior vagina about one and a half inches from the cervix. The posterior portion projected back into the pelvis and over the urethral orifice and was excoriated with urine. The anterior part, by its weight had drawn the anterior vagina well over the pelvic brim and into the abdominal cavity.

Length.—9 inches.

Depth.—3½ inches at its broadest part.

Breadth.—3 inches.

Weight.—2 lb. 5½ oz.

Remarks.—It is interesting to note that only three days of discomfort was recorded and the subject appeared bright all the time. Reconsidering the case and in light of the post-mortem findings, one wonders if the growth could have been successfully removed piecemeal from a mid-line incision. It was impossible from a flank incision or from the vagina, in view of its size. However, a full bladder would have complicated the mid-line incision. The attachment of the growth to the vaginal wall was very small but this was not recognised until post-mortem. I think that removal would have been very difficult, and hazardous to have attempted.

Abstracts of Current Literature

Seasonal Variation in the Semen of Bulls. By R. W. PHILLIPS, *et alia* (1943). *Amer. J. Vet. Res.* Vol. *4* (No. 1), 115-119.

SEMEN from three beef Shorthorn and three dairy Shorthorn bulls was studied at intervals of two weeks throughout a year. Semen samples were collected by artificial vagina; observations were made on motility, volume, number of sperm per c.c., total number of sperm, and the proportions of abnormal heads, necks, middle pieces, tails, and total abnormal spermatozoa.

Seasonal trends in many of the factors studied could be observed. The volume of semen produced was highest in the winter and lowest in the summer. Total sperm produced was highest in the spring and lowest in the fall. Abnormal heads, necks and middle pieces were highest in the summer months and lowest in the winter. Abnormal tails had the opposite trend, being highest in the

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winter and lowest in the summer. No explanation for this trend of abnormal tails is evident, but it is suggested that the cold weather had an effect on the tails after the collections were made.

An attempt was made to evaluate the importance of the various sources of variation in semen quality. By the statistical method used, an estimate is obtained of the percentage of variation to each of the various sources. In general, variations between samples within bulls and season was the source of the greater variation, i.e., the semen from a bull from successive services varies more than any other factor in most cases. In motility score, differences between bulls and variations between samples account for nearly 86 per cent. of the total variation. In volume of semen produced, the greatest variation was between breeds and between samples. Variations between samples and between bulls accounted for most of the variation in sperm count, though there was a definite seasonal trend. In abnormal sperm, differences between bulls, seasons, and samples account for the major portion of the variance. There was little seasonal effect on the hours of survival of sperm in storage. Aside from variations between samples, it may be said that differences between bulls had the greatest effect on semen quality and that the main seasonal effect was noticed in semen morphology.

Examination of the results of 1,135 meetings over a seven-year period revealed that the highest percentage of fertile matings occurred in April (59.6 per cent.) and the lowest in August (40.8 per cent.). It appears that the decrease noted in certain measures of semen quality during the summer months is reflected in decreased breeding efficiency in the herd. This is probably due to the effects of a warm environment on the bull and on his semen production. (A similar decline in the fertility of rams during the hot season has also been observed in the U.S.A. and in Australia.)

Vitamin A Deficiency Not the Cause of Joint Lesions in Horses. By GEORGE H. HART, HAROLD GOSS and HAROLD R. GUILBERT. *Amer. J. Vet. Res.* Vol. *4* (No. 11), April, 1943.

IN 1941, Howell, Hart, and Ittner published the results of experiments in which ten horses were allowed to succumb from lack of vitamin A after being on the deficient diets from 286 to 1,139 days. Accompanying the vitamin symptoms in all cases was joint involvement, with lameness and with characteristic rarefying lesions in the joint cartilages. The data made it necessary to conclude: "Positive evidence that the joint condition was due primarily to the vitamin A deficiency was not obtained, but the evidence pointed to the possibility." Because of the importance of arthritis in animals and particularly in human beings, we began experiments with four additional horses in order to ascertain the relation of vitamin A to these joint lesions.

Rations.—After the first two weeks, the ration used contained no roughage. During this period, some straw was used to overcome looseness of the bowels, which occurred in changing to the experimental rations. For both groups the feed was identical, being taken from the same mix and bin throughout the experiment.

In addition, the animals in group 1 received twice weekly 5 c.c. of high-potency shark-liver oil. (Horses Nos. 1 and 2.) Group 2 (Nos. 3 and 4) received no oil.

The ration was made from: Dried molasses beet pulp, 642 lb., 53.5 per cent.; rolled barley, 120 lb., 10 per cent.; rolled oats, 120 lb., 10 per cent.; wheat bran, 120 lb., 10 per cent.; linseed meal, 120 lb., 10 per cent.; cottonseed meal, 60 lb., 5 per cent.; bone meal, 18 lb., 1.5 per cent. The Ca-P ratio of the ration averaged 1.8 to 1.

Symptoms.—At various times during the experiment lameness was noted in both groups.

Joint lesions were not suspected in both groups until post-mortem, when their presence was recognised to have caused some of the lameness.

In general the amount of the lameness compared with the extent of the joint lesions was very slight. It would probably have become more manifest had the animals been obliged to do normal work. No. 4 was the only animal to die naturally.

Post-mortem examination.—The blood of the horses in group 1 remained high in vitamin A throughout, whereas the blood of those of group 2 showed only a trace at the time of death. At the autopsy, pieces of liver were saved in all four cases, and the vitamin A content determined. Horse No. 1 showed 1,173 I.U., and No. 2, 1,115 I.U. per gm. of liver tissue, whereas none was detected in the liver tissue of either Nos. 3 or 4.

The only macroscopic finding of note in the post-mortem examination of these horses was the joint lesions. These were present to an almost equal extent in all four animals. They consisted of the same rarefying condition found in the animals reported in the previous work.

In the humero-radial and tibio-tarsal joints, the pronounced ulceration of the cartilages duplicated upon opposing articular surfaces was characteristic. This same alteration was present to a greater or lesser degree in all the major joints. No exostoses occurred on the bones of any of these horses.

Discussion

This experiment was staged primarily to clear up the relation of vitamin A to the presence of the joint lesions. It proved conclusively that vitamin A is not the causative factor. It quite definitely demonstrated that low vitamin A intake carried to a fatal termination did not seriously affect the blood levels of vitamin C.

The rations were so made up that the animals were protected from possible deficiency of vitamin D. Since the calcium-phosphorus intake was ample, and since the ratio throughout the experimental period averaged 1.8 to 1, these important mineral elements could not be considered in any way connected with the causation of the lesions.

Discussion of other known substances, such as the cartilage factor, the green-grass factor, and possible yet undetermined factors, is of little point without supporting data. The substances lacking in this concentrate diet that resulted in the joint changes, if they are of dietary origin, would probably be supplied by green grass or good quality leafy hay. To substantiate this hypo-

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thesis, additional experimentation would be necessary. It would also be desirable to know how soon dietary limitations could produce these changes, and whether, when well established, they could be cured by improved dietary regimen. We doubt that complete regeneration ever occurs once the degenerative process has become definitely established. Hence their presence, post-mortem, would be no definite criterion as to when they had developed in the life span of the particular animal.

As this paper was about to go to press, the authors' attention was called to the elaborate work of Bennett, Waine, and Bauer[7] on changes in the knee joint at various ages in man. They agree with a long list of modern investigators who believe that the initial anatomically demonstrable lesion in man is a degenerative process in the superficial layer of articular cartilage. The finding of lesions in markedly different stages of development in the affected horses coming to post-mortem examination in our work presented ample evidence that this is also the case in the horse.

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Review

The Bloodstock Breeders' Review. The British Bloodstock Agency, Ltd., 26, Charing Cross Road, London, W.C.2. Vol. XXXI. 1942. Price: 40s. net.

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